beta adrenergic blockade. In the present study, cardiac performance in the dog heart-lung preparation was improved by MJ 1988. In both the heart-lung preparation and in the intact, anesthetized dog, the positive myocardial inotropic effect was accompanied by increases in coronary blood flow, pulmonary vasodilation and bronchodilation. With much larger doses, cardiac depression and arrhythmias were produced, but the wide margin of safety between therapeutic and toxic doses indicates this compound to be of possible utility in the treatment of cor pulmonale. (*Aviado, D. M., and others: The Cardiopulmonary Effects of Quinazoline (MJ 1988): Cardiac Stimulant, Pulmonary Vasodilator and Bronchodilator, J. Pharmacol. Exp. Ther. 155: 76 (Jan.) 1967.*)

CARDIAC TAMPONADE Injection of 75 per cent Hypaque via a catheter into the pericardial sac of dogs results in an accumulation of fluid within the sac far in excess of the volume originally injected. The explanation for the increase of fluid is that Hypaque has an osmolarity eight times that of serum and causes fluid to be drawn into the pericardial sac. The clinical implication of this study is if while performing cardiac catheterization and cinecardioangiography, a quantity of the contrast material is accidently deposited in the pericardial sac, pericardial tamponade occurring within one hour might be expected. (*Popper, R. W., and others: Cardiac Tamponade due to Hypertonic Contrast Medium in the Pericardial Sac Following Cineangiography, Circulation 35: 933 (May) 1967.*)

PULMONARY CIRCULATION The effect of an acute increase in blood hydrogen ion concentration (*p*H fall 0.07 units) upon pulmonary circulation was studied in patients with obstructive lung disease. Pulmonary artery systolic and diastolic pressures rose 5 and 2 mm. of mercury, respectively, with no change in wedge pressure, cardiac output, or pulmonary blood volume. The most marked pressure elevations were seen in patients with a hemoglobin saturation less than 91 per cent. The changes observed are attributed to pulmonary vasoconstriction rather than increased pulmonary blood volume or left sided heart failure. (*Harvey, R. M., and others: Further Observations on the Effect of Hydrogen Ion on the Pulmonary Circulation, Circulation 35: 1019 (June) 1967.*)

PULMONARY VESSELS Histologic findings from lung biopsies taken at the time of open heart surgery are correlated with clinical and hemodynamic data. Patients were operated on for correction of either atrial septal defect (ASD), ventricular septal defect (VSD), or patient ductus arteriosus (PDA). In the ASD group, pulmonary vessels did not differ from controls. Within the ASD group there was no difference between high and low flow. In the VSD group there was an increase in medial and intimal thickness which correlated with increased pulmonary artery pressure but not flow. Pulmonary hypertension may provoke arterial lesions and, in turn, be perpetuated by them. An increased flow in itself has little effect upon pulmonary vasculature morphology. (*Wagenvoort, C. A., and others: Effect of Flow and Pressure on Pulmonary Vessels, Circulation 35: 1028 (June) 1967.*)

Respiration

AIRWAY TONE A variety of techniques were used to study effects of changes in blood flow and alveolar P_{O_2} and P_{CO_2} on bronchomotor tone in dogs. Increase in blood flow to an isolated, ventilated and perfused lobe caused bronchodilitation by increasing P_{CO_2} in alveolar gas. Increase in inhaled P_{CO_2} to the isolated lobe produced a decrease in bronchomotor tone while systemic hypercarbia produced bronchoconstriction mediated via the vagus. Local hypoxia caused bronchoconstriction indirectly by decreasing P_{CO_2} while systemic hypoxia produced increased vagal activity with bronchoconstriction. All of these responses are important in the regulation of ventilation and perfusion to a lobe. (*Samanek, M., and Aviado, D. M.: Interrelationships Between Pulmonary Blood Flow and Bronchomotor Tone: P_{O_2} and P_{CO_2}, J. Appl. Physiol. 22: 719 (April) 1967.*)